Active Trigger Points in the Cervical Musculature Determine Altered Activation of Superficial Neck and Extensor Muscles in Women with Migraine

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ABSTRACT

Objective: Previous studies have demonstrated the presence of active TrPs in women with migraine reproducing their headache attacks. No study has investigated if these TrPs can alter muscle function in the cervical spine in migraine. Our objective was to analyze differences in activation of superficial neck flexor and extensor muscles in women with migraine considering the presence of active trigger points (TrP) in splenius capitis (SC), upper trapezius (UT), and sternocleidomastoid (SCM) muscles. **Methods**: Surface EMG was recorded from superficial flexor (SCM and anterior scalene) and extensor (SC) muscles bilaterally as subjects performed a staged task of cranio-cervical flexion (CCF; 5 contractions representing a progressive increase in CCF range of motion) in 70 women with migraine. They were stratified according to presence or absence of active TrPs in SCM, SC or UT musculature. Comparison of normalized root mean square (RMS) values was conducted with 2x5 ANCOVA with task level as the within-subject variable, group stratified by active TrPs as the between-subjects variable and the presence of neck pain as a co-variable. **Results:** All patients exhibited active TrPs in their cervical muscles which reproduced their migraine. Women with migraine exhibiting active TrPs in the SCM ($P<0.01$), UT ($P<0.05$) or SC ($P<0.05$) muscles had lower normalized RMS values of their superficial neck flexors than those without active TrPs in the same muscles. In addition, subjects exhibiting active TrPs in the SC and UT (both, $P<0.05$) muscles had higher normalized RMS values in the SC muscle than those without active TrPs in the same muscles. **Conclusion:** The presence of active TrPs in the cervical musculature determines altered activation of superficial neck and extensor muscles during low-load, isometric CCF contractions in women with migraine.

Key words: migraine, cranio-cervical flexion test, trigger points, electromyography.

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INTRODUCTION

Migraine is a disabling primary headache described as a chronic disorder with recurrent attacks. Migraine has worldwide prevalence ranging from 5 to 12%.[1] Although migraine pain is mostly perceived in the ophthalmic distribution of the trigeminal nerve, neck pain is also a prevalent concomitant symptom in this population.[2-4] In fact, approximately 76% of migraine patients also report the presence of neck pain,[5] which can occur as a premonitory manifestation, during the headache phase or even in the interictal period.[6]

It has been suggested that the association between neck pain and migraine occurs because the trigeminal-cervical convergence provides an anatomical and neurophysiological path for interaction via the convergence of cervical and trigeminal nociceptive afferents in the trigemino-cervical nucleus caudalis.[7,8] In addition, central sensitization presenting in most individuals with migraine may facilitate neck pain and related disorders.[9] The presence of neck pain has a negative influence on migraine by reducing the pharmacological treatment response.[10,11]

Experience of pain or even the anticipation of pain may promote a variety of motor control changes involving redistribution of activity within and between muscles.[12] Previous studies investigating neck muscle activity in patients with migraine revealed varying results. For instance, during maximal voluntary isometric contractions of the neck musculature, an increased co-activation of antagonist muscles was observed in girls[13] and women with either episodic or chronic migraine[14] while maximal strength seems to be affected only in women with chronic migraine.[14] However, during low-load tasks such as the cranio-cervical flexion test (CCFT) no significant differences in

activation of superficial neck flexors were observed in individuals with migraine.[15,16] These varying results may reflect the different tasks examined (strength versus motor control) but may also suggest that changes in the activation the cervical musculature are only present in some patients with migraine, rather than being unequivocally associated with most migraine patients.

Interestingly, these previous studies have not taken into account the presence of trigger points (TrPs) in the neck musculature. Yet, it is well described that patients with migraine exhibit more active TrPs, those which reproduce the migraine attack when stimulated,[17] in the cranio-cervical muscles compared to subjects without headache.[18,19] The presence of TrPs has been associated with motor disturbances as they can promote fatigue, altered coordination, and altered pattern of intramuscular activity.[20-22]

No previous study has investigated the potential influence of active TrPs in the neck musculature on electromyographic activity of superficial neck flexor and extensor muscles during the CCFT in individuals with migraine. Therefore, the aim of the current study was to investigate differences in activation of superficial neck flexor and extensor muscles during the CCFT in migraine patients considering the presence of active TrPs in splenius capitis (SC), upper trapezius (UT) and sternocleidomastoid (SCM) muscles. We hypothesized that the presence of active TrPs in the cervical musculature would be associated with altered activity of the superficial neck muscles.

METHODS

Participants

Patients with migraine without aura were recruited from an urban regional hospital between November 2014 and October 2015. Patients were diagnosed following the third edition of International Headache Society criteria by an experienced neurologist.[23] Migraine features including location, quality of pain, years with disease, the frequency and intensity of attacks, family history and medication intake were collected as clinical history. No abnormalities were detected in routine blood analyses with ESR or urine analyses. An X-ray examination of the skull and cervical spine and a CT scan or MRI of the head were invariably performed, and did not show any structural lesion. They were excluded if they presented any of the following criteria: 1, other concomitant primary or secondary headache; 2, medication overuse headache; 3, history of cervical or head trauma (i.e., whiplash); 4, pregnancy; 5, history of cervical herniated disk or cervical osteoarthritis on medical records; 6, any systemic degenerative disease, e.g., rheumatoid arthritis, lupus erythematous; 7, diagnosis of fibromyalgia syndrome; 8, anesthetic block in the past 3 months; or, 9, receiving physical therapy intervention in the head and neck the previous 6 months. A careful clinical examination of each participant was conducted to determine inclusion and exclusion criteria.

All participants signed the informed consent form before their inclusion in the study. The local Ethics Committee of Hospital Rey Juan Carlos (HRJ 07/14) approved the study design.

Clinical measures

Clinical data including years with migraine, migraine frequency (days per month), intensity of pain attacks (numerical pain rate scale, 0-10), headache duration (hours per

attack), as well as presence of self-reported neck pain, including report of the frequency, intensity and years with neck pain were systematically collected.

Cranio-cervical flexion test (CCFT)

The CCFT is a low-load graded test of deep cervical flexor muscle performance with five progressive stages guided by a pressure biofeedback unit (Stabilizer®, Chattanooga Group Inc. South Pacific, USA, **Fig. 1**). It is performed with the subject in supine, with the head and neck in a neutral position. The pressure biofeedback unit is placed behind the subject's neck in the suboccipital region, with an initial inflation pressure of 20 mmHg.[24]

First, participants were familiarized with the test. Subjects were instructed to perform a gentle head-nodding action of cranio-cervical flexion over five incremental stages of increasing range of motion (2 mmHg each stage) and each stage was maintained for 10 seconds. Head extension, head lift or opening the mouth, described as compensations strategies,[24] were discouraged at familiarization time.

After the familiarization phase, a rest period of 1 minute was permitted. Subjects then performed the CCFT by holding each target level for 10s with 30s rest between levels. During the holding phase, surface electromyography of selected neck flexors and extensor muscles was acquired. The full CCFT was repeated twice with a 15min rest between. All subjects performed all CCFT levels and compensatory strategies were not controlled during the formal test. The CCFT examination was conducted by an assessor blinded to the presence or absence of TrPs.

Electromyography (EMG) acquisition and processing

After gentle skin abrasion using abrasive paste, bipolar surface EMG was recorded with pairs of electrodes positioned 20mm apart (Ambu®-Blue Sensor N-50-K/25) and firmly fixed with adhesive tape bilaterally over the following cervical muscles: 1, the

158 sternal head of SCM muscle, over the muscle belly at1/3 of the distance from the sternal
159 notch to the mastoid process;[25] 2, anterior scalene (AS): over the muscle belly parallel to
160 the clavicular head of the SCM;[25] and, 3, SC muscle: over the muscle belly at C2-C3
161 level between the uppermost parts of the SCM and UT muscles.[26] The reference
162 electrode was placed on the wrist of the participants. Myoelectric signals from SCM,
163 AS, SC and UT muscles were amplified by 5000 (EMG16, 16-channel amplifier,
164 LISiN-OT Bioelettronica®, Torino, Italy), filtered (-3dB bandwidth, 10-450 Hz),
165 sampled at 2048 Hz, and converted to 12-bit digital samples.

166 Customized MATLAB code (The Mathworks™, Natick, MA, USA) was used
167 for data processing. EMG raw signals were band-filtered at a 20-400Hz (4th order
168 Butterworth) and the average Root Mean Square (RMS) was calculated from each 10 s
169 contraction. Neck flexor and extensor RMS values were normalized and expressed as a
170 percentage of the maximum RMS value during a reference voluntary contraction. The
171 reference activity for superficial neck flexors was a head lift task, and for superficial
172 neck extensors was head extension against the table in the supine position. For analysis
173 purposes, the mean RMS values were averaged over the two repetitions for each CCFT
174 stage. Finally, the mean of both sides right and left, for each muscle were considered in
175 the analysis for all CCFT stages.

176 **Trigger Point Identification**

177 Screening for TrPs was performed by an assessor with 6 years of experience in
178 TrP diagnosis. The SCM, SC and UT muscles were assessed bilaterally since TrPs in
179 these muscles referred pain to the head mimicking migraine.[18,19] TrP diagnosis was
180 performed according the following criteria:[17] 1, presence of a palpable taut band in the
181 muscle; 2, presence of a painful spot in the taut band; 3, local twitch response on
182 snapping palpation of the taut band; and, 4, reproduction of referred pain during manual

examination. TrP diagnosis was conducted using snapping palpation (first to locate the taut band, and then moving the thumb tip back and forth to roll the underlying fibers) to induce a local twitch response, and flat palpation (placing the padded aspect of the thumb on the painful spot and applying pressure against the underlying tissue or bone) to induce the referred pain.

Participants were evaluated during interictal migraine states and pain-free states, and when at least one week had elapsed since the last migraine attack to avoid migraine related allodynia. TrPs were considered active when the referred pain elicited during manual examination reproduced the migraine attack features that the subject usually suffered from, and, therefore, the pain was recognized as a familiar pain.[17] Patients were classified as having active TrPs when they had TrPs reproducing their migraine attack in at least one muscle, either left or right side.

Statistical analysis

Data were analyzed with SPSS software version 20.0 (SPSS Inc©, Chicago, IL). Means and 95% confidence intervals (95%CI) were calculated for the clinical variables. Patients were stratified according to the presence of active TrPs in the SCM, SC, and UT muscles separately. The comparison for the normalized RMS values was conducted with a 2x5 analysis of co-variance (ANCOVA) with CCFT stage (22 mmHg, 24 mmHg, 26 mmHg, 28 mmHg, and 30 mmHg) as the within-subject variable, and stratification (presence or absence of active TrPs) as the between-subject variable and the presence of neck pain as co-variate. Separates ANCOVAs were conducted depending on the muscle affected by active TrPs (SCM, SC and UT). The statistical analysis was conducted at 95% confidence level. A P value < 0.05 was considered statistically significant.

RESULTS

Clinical features of the sample

From 100 eligible subjects with migraine who accepted to participated, 30 were excluded for the following reasons: other co-morbid headaches (n=15), receiving anesthetic block (n=6) or botulinum toxin A (n=6) in the past 3 months, and reporting previous head or neck trauma (n=3). Finally, 70 women, mean age: 42±12 years old, with episodic migraine without aura were included. A total of 58 women (83%) self-reported neck pain. All women exhibited active TrPs reproducing their migraine attacks. The mean ± SD number of active TrPs for each patient with migraine was 3.0± 1.5. The UT muscle was the most affected by active TrPs in our sample (n=41, 59%). **Table 1** summarizes demographic and clinical data of the total sample. The clinical status of patients was not dependent on the presence of TrPs in each cervical muscle (**Table 2**).

Neck flexor activity and TrPs

Normalized RMS values for SCM and AS muscles during the five stages of the CCFT in those patients with active TrPs in the sternocleidomastoid, upper trapezius and splenius capitis are shown in **Figs. 2-4**. There was an increase in EMG amplitude of the SCM and AS with the progressive stages of the test independently of the presence of active TrPs in the SCM muscle (SCM: $F=16.57$; $P<0.001$, AS: $F=15.35$; $P<0.001$), upper trapezius (SCM: $F=12.59$; $P<0.001$, AS: $F=16.54$; $P<0.001$), or SC muscle (SCM: $F=16.15$; $P<0.001$, AS: $F=10.18$; $P<0.001$). Women with migraine exhibiting active TrPs in the SCM muscle (SCM: $F=10.307$; $P=0.002$, AS: $F=7.169$; $P=0.009$), UT muscle (SCM: $F=5.19$; $P=0.026$, AS: $F=4.491$; $P=0.044$) or SC muscle (SCM: $F=7.852$; $P=0.007$, AS: $F=6.437$; $P=0.018$) showed lower normalized RMS values of their superficial neck flexors than those without active TrPs in the same muscles (**Figs. 2-4**). The presence of neck pain did not influence the results (SCM: $P>0.253$, AS: $P>0.356$).

Neck extensor activity and TrPs

Normalized RMS values for SC muscle during the five stages of the CCFT in those patients with active TrPs in the SCM, UT and SC are shown in **Fig. 5**. There was also an increase in EMG amplitude of the SC with the progressive stages of the test independently of the presence of active TrPs in either the SCM ($F=4.41$; $P=0.039$), UT ($F=4.591$; $P=0.045$), or SC ($F=11.176$; $P<0.001$) muscles. In contrast to the results for the flexor muscles, the results revealed higher normalized RMS values in the SC muscle in women with migraine exhibiting active TrPs in the SC ($F=4.05$; $P=0.046$) and UT ($F=4.014$; $P=0.046$) muscles (**Fig. 5**) compared to those without active TrPs in the same muscles. No significant differences were observed for normalized RMS values in the SC in those patients with active TrPs in the SCM muscle ($F=0.290$; $P=0.592$, **Fig. 5**). The presence of neck pain (SC: $P>0.213$; UT: $P>0.293$) did not influence the results.

DISCUSSION

Women with migraine exhibiting active TrPs in the SCM, SC and UT muscles had lower activation of their superficial neck flexors, i.e., SCM and AS, during low-load CCF contractions. In addition, the presence of active TrPs in the superficial neck extensors, i.e., SC and UT, determined increased activation of the SC muscle during cranio-cervical flexion contractions.

It is well known that noxious stimulation of a muscle, e.g. with experimental muscle pain via intramuscular injection of hypertonic saline, induces a temporary decrease of EMG amplitude of the painful muscle together with compensatory strategies within the same muscle[27,28] or across synergistic muscles.[29-31] It may be speculated that a long-lasting nociceptive irritant, such as an active TrP, also induces inhibition of the painful muscle when activated. This knowledge may explain the reduced activation of the SCM

and AS muscles in individuals with active TrPs in the same musculature. Interestingly, reduced activation of the SCM and AS was also noted in women with active TrPs in the SC or UT muscles which implies that the altered muscle strategy is not necessarily due to pain induced inhibition locally within the muscle.

The observation of reduced activation of the SCM and AS during the CCFT in the women with migraine and active TrPs is in contrast to observations in people with primary neck disorders, including cervicogenic headache.[15,16] Rather, people with cervical spine disorders show higher activity of the SCM and AS muscles during the CCFT which has been shown to be an indicator of poor performance of the deep neck flexor muscles, i.e., longus colli and longus capitis.[32,33] However, migraine is a primary headache mainly associated to brain dysfunction with deficient regulation of excitatory-inhibitory balance during cortical activity leading to trigemino-vascular sensitization. Thus, although individuals with migraine usually suffer from concomitant neck pain,[5] they do not have a primary neck pain disorder which would explain these contrasting results. Nevertheless, we observed that the presence of active TrPs within the cervical musculature implies different activation of the neck flexor muscles compared to those without active TrPs in the same muscles. Interestingly, differences in muscle activation was associated to the presence or absence of active TrPs, but not related to the presence of neck pain in our study.

During tasks with low mechanical demands, performance can be maintained despite pain, also via modification of antagonist musculature activity.[30,34] Indeed, one theory of the motor adaption to pain indicates that muscle pain induces reorganization of the motor strategy characterized by reduced activity of agonist muscles and increased activity of antagonist muscles (pain adaptation theory).[35] The current work supports the observation of increased antagonist muscle activity since increased SC muscle activity

was noted when active TrPs were present within the SC or UT muscles. In support of the current findings, individuals with chronic, but not episodic, migraine exhibit higher activity of their superficial neck extensors (i.e., SC muscle) during low-load, isometric cranio-cervical flexion contractions compared to non-headache individuals (unpublished observations) and women with chronic tension type headache also show greater co-activation of antagonist muscles (i.e. the SC muscle) during isometric neck flexion contractions compared with headache-free subjects.[36] Thus, increase co-activation of antagonist musculature appears to be a common feature in people with headache. The results from the current study suggest that increased antagonist muscle co-activation is even more likely in those with active TrPs.

Overall, the observation that TrPs are associated with changes in the activation of agonist and antagonist muscles is consistent with earlier findings. Ibarra et al observed increased muscle activity at latent TrPs in an antagonist muscle (i.e., posterior deltoid muscle) during shoulder flexion task.[22] Lucas et al[37] found that the presence of latent TrPs impaired recruitment or timing of muscle activation when performing active joint movement and Ge et al[20] found that the presence of latent TrPs induced incoherent muscle activation patterns in synergist musculature during muscle contractions. However, these studies included latent TrPs, but not active TrPs, which limit the clinical relevance of their data since latent TrPs are not related to clinical pain complaints. Our study is the first one showing that the presence of active TrPs was associated with a different pattern of agonist and antagonist muscle activation in patients with headache. Our finding has potential implication for clinical practice. Since the presence of active TrPs in the cervical musculature is related to altered pattern of neck muscle activation, it would be recommended that clinicians first treat these TrPs before start any therapeutic

exercise program targeting at normalizing motor control disturbances observed in these patients.

Although the study expands current knowledge on changes in muscle behavior in individuals with migraine, potential limitations should be recognized. First, we only included women with migraine, therefore, we do not know if the same results would be observed in men. Second, we included a single low-load cranio-cervical flexion task for investigating muscular activity, but this task does not necessarily represent muscle demands during daily life activities. Third, psychological features, e.g. fear of movement, were not measured and may have proven useful in understanding the mechanisms underlying the observed altered muscle behavior in people with migraine. Further, a control group of headache-free individuals was not included; thus, although we can confirm differences in the activation of the neck musculature between women with and without active TrPs in their cervical muscles, we cannot confirm that the changed pattern of activation within the migraine group with active TrPs would be significantly different to asymptomatic people.

CONCLUSION

In the current study, all women with migraine exhibited active TrPs in the neck muscles reproducing their migraine attack. Women with migraine who have active TrPs in the cervical musculature show an altered pattern of neck muscle activation during a low-load cranio-cervical contraction compared to those without active TrPs in the evaluated muscle. Alterations of afferent input (i.e., painful stimulus induced by active TrPs) appear to influence muscle activation at a multi-muscular level.

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Legend of Figures

Figure 1: Pressure biofeedback unit (Stabilizer®, Chattanooga Group Inc. South Pacific, USA) used during the cranio-cervical flexion text (CCFT)

Figure 2: The normalized root mean square (RMS) values for the sternocleidomastoid and anterior scalene muscles for the five stages of the cranio-cervical flexion test depending on the presence or absence of active trigger points (TrPs) in the sternocleidomastoid muscle (SCM - yes, n=36 / no, n=41). Values for the left and right muscles have been averaged. Data are expressed as means and SEM. * $P<0.05$; ** $P<0.01$

Figure 3: The normalized root mean square (RMS) values for the sternocleidomastoid and anterior scalene muscles for the five stages of the cranio-cervical flexion test depending on the presence or absence of active trigger points (TrPs) in the upper trapezius muscle (UT- yes, n=41 / no, n=29). Values for the left and right muscles have been averaged. Data are expressed as means and SEM. * $P<0.05$; ** $P<0.01$

Figure 4: The normalized root mean square (RMS) values for the splenius capitis muscle for the five stages of the cranio-cervical flexion test depending on the presence or absence of active trigger points (TrPs) in the splenius capitis muscle (SC - yes, n=29 / no, n=41). Values for the left and right muscles have been averaged. Data are expressed as means and SEM. * $P<0.05$; ** $P<0.01$

Figure 5: The normalized root mean square (RMS) values for the splenius capitis muscle for the five stages of the cranio-cervical flexion test depending on the presence or absence of active trigger points (TrPs) in the sternocleidomastoid (SCM - yes, n=36 / no, n=41), upper trapezius (UT- yes, n=41 / no, n=29), or splenius capitis (SC - yes, n=29 / no, n=41) muscles. Values for the left and right muscles have been averaged. Data are expressed as means and SEM. * $P<0.05$; ** $P<0.01$